

08/765,026

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Paper # 16

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14 and "superoxide dismutase\$"	7

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14 and "superoxide dismutase\$"

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Entry 1 of 7

File: USPT

Oct 5, 1999

US-PAT-NO: 5962478

DOCUMENT-IDENTIFIER: US 5962478 A

TITLE: Inhibition of tumor necrosis factor .alpha.

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KWIC	Image
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2. Document ID: US 5929042 A

Entry 2 of 7

File: USPT

Jul 27, 1999

US-PAT-NO: 5929042

DOCUMENT-IDENTIFIER: US 5929042 A

TITLE: Antisense compounds which prevent cell death and uses thereof

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KWIC	Image
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3. Document ID: US 5871729 A

Entry 3 of 7

File: USPT

Feb 16, 1999

US-PAT-NO: 5871729

DOCUMENT-IDENTIFIER: US 5871729 A

TITLE: Superoxide dismutase-4

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KWIC	Image
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4. Document ID: US 5834306 A

Entry 4 of 7

File: USPT

Nov 10, 1998

US-PAT-NO: 5834306

DOCUMENT-IDENTIFIER: US 5834306 A

TITLE: Tissue specific hypoxia regulated therapeutic constructs

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KWIC	Image
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5. Document ID: US 5770443 A

Entry 5 of 7

File: USPT

Jun 23, 1998

US-PAT-NO: 5770443

DOCUMENT-IDENTIFIER: US 5770443 A

TITLE: Apoptosis-modulating proteins, DNA encoding the proteins and methods of use thereof

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KMC	Image
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6. Document ID: US 5571797 A

Entry 6 of 7

File: USPT

Nov 5, 1996

US-PAT-NO: 5571797

DOCUMENT-IDENTIFIER: US 5571797 A

TITLE: Method of inducing gene expression by ionizing radiation

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KMC	Image
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7. Document ID: US 5506133 A

Entry 7 of 7

File: USPT

Apr 9, 1996

US-PAT-NO: 5506133

DOCUMENT-IDENTIFIER: US 5506133 A

TITLE: Superoxide dismutase-4

Full	Title	Citation	Front	Review	Classification	Date	Reference	Claims	KMC	Image
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Terms	Documents
14 and "superoxide dismutase\$"	7

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Document Number 7

Entry 7 of 7

File: USPT

Apr 9, 1996

DOCUMENT-IDENTIFIER: US 5506133 A

TITLE: Superoxide dismutase-4

BSPR:

This invention relates to newly identified polynucleotides, polypeptides encoded by such polynucleotides, the use of such polynucleotides and polypeptides, as well as the production of such polynucleotides and polypeptides. More particularly, the polypeptide of the present invention is Superoxide Dismutase-4 (SOD-4).

BSPR:

Two of these, O.sub.2.sup.- and OH.sup.-, have single unpaired electrons and are therefore called free radicals. A few percent of the oxygen consumption in the body has been estimated to lead to the formation of the toxic reduction intermediates, The toxic affects of oxygen are mainly ascribable to the actions of these intermediates,

BSPR:

Organisms living in the presence of oxygen have been forced to develop a number of protective mechanisms against the toxic oxygen reduction metabolites. The protective factors include superoxide dismutases (SOD) which dismutate the superoxide radical and are found in relatively constant amounts in mammalian cells and tissue. The best known of these enzymes is CuZnSOD which is a dimer with a molecular weight of 33,000 containing two copper and two zinc atoms. CuZnSOD is found in the cytosol and in the intermembrane space of the mitochondria. MnSOD is a tetramer with a molecular weight of 85,000 containing four Mn atoms, and is mainly located in the mitochondrial matrix. Until recently the extra cellular fluids were assumed to lack SOD activity. However U.S. Pat. No. 5,248,603 recently disclosed the presence of a superoxide dismutase in extracellular fluids (e.g., blood plasma, lymph, synovial fluid and cerebrospinal fluid) which was termed EC-SOD.

BSPR:

Mutations in the CuZnSOD gene occur in patients with the fatal neurodegenerative disorder familial amyotrophic lateral sclerosis. Screening of the CuZnSOD coding region revealed that the mutation Ala 4 to Val in exon 1 was the most frequent one, mutations were identified in exons 2, 4 and 5 but not in the active site region formed by exon 3. Thus, defective CuZnSOD is linked to motor neuron death and carries implications for understanding and possible treatment of familial amyotrophic lateral sclerosis. The polypeptide of the present invention, SOD-4, is structurally and functionally related to CuZnSOD.

BSPR:

Japanese Patent No. 4248984 discloses a superoxide dismutase derivative which has a longer half-life in blood than SOD and therefore helps treat various diseases.

BSPR:

Japanese Patent No. 2156884 discloses a 153 amino acid polypeptide having human superoxide dismutase properties and a DNA sequence encoding

such polypeptide, a DNA sequence expressed by the nucleic acid sequence and production of the polypeptide by culture of host cells.

BSPR:

Japanese Patent No. 63313581 discloses a pharmacologically active modified superoxide dismutase which is obtained by reacting SOD with a compound containing an amino or carboxyl group.

DEPR:

The polynucleotides of the present invention may be employed for producing polypeptides by recombinant techniques. Thus, for example, the polynucleotide may be included in any one of a variety of expression vectors for expressing a polypeptide. Such vectors include chromosomal, nonchromosomal and synthetic DNA sequences, e.g., derivatives of SV40; bacterial plasmids; phage DNA; baculovirus; yeast plasmids; vectors derived from combinations of plasmids and phage DNA, viral DNA such as vaccinia, adenovirus, fowl pox virus, and pseudorabies. However, any other vector may be used as long as it is replicable and viable in the host.

DEPR:

Transcription of the DNA encoding the polypeptides of the present invention by higher eukaryotes is increased by inserting an enhancer sequence into the vector. Enhancers are cis-acting elements of DNA, usually about from 10 to 300 bp that act on a promoter to increase its transcription. Examples including the SV40 enhancer on the late side of the replication origin bp 100 to 270, a cytomegalovirus early promoter enhancer, the polyoma enhancer on the late side of the replication origin, and adenovirus enhancers.

DEPR:

If the supply of blood to a tissue is cut off, the tissue will slowly become necrotic. Oxygen radicals formed as a result of the reappearance of oxygen in previously ischaemic tissue appear to contribute to the damage. Thus the removal of these free radicals by SOD-4 helps to protect tissue against damage. SOD-4 may be employed to reduce the incidence of ischaemia and reperfusion induced arrhythmias by a similar mechanism, since SOD proteins have been reported to affect these conditions (Woodward, B. et al, J. Mol. Cell. Cardiol. 17:485-493 (1985). In the same manner, SOD-4 may be employed to treat cerebral ischaemia and kidney ischaemia, SOD proteins have been demonstrated to protect tissues in ischaemia or anoxiareperfusion models in the kidney (Baker, G. L., et al., Am. Surg., 202:628-41 (1985).

DEPR:

Similarly, cells may be engineered in vivo for expression of a polypeptide in vivo by, for example, procedures known in the art. As known in the art, a producer cell for producing a retroviral particle containing RNA encoding the polypeptide of the present invention may be administered to a patient for engineering cells in vivo and expression of the polypeptide in vivo. These and other methods for administering a polypeptide of the present invention by such method should be apparent to those skilled in the art from the teachings of the present invention. For example, the expression vehicle for engineering cells may be other than a retrovirus, for example, an adenovirus which may be used to engineer cells in vivo after combination with a suitable delivery vehicle.

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Terms	Documents
19 and superoxide	4

Database: [All Databases \(USPT + EPAB + JPAB + DWPI + TDBD\)](#)

Refine Search:

19 and superoxide

Search History

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ALL	16 and neuropatholo\$	48	<u>L7</u>
ALL	adenovirus\$	5997	<u>L6</u>
ALL	14 and "superoxide dismutase\$"	7	<u>L5</u>
ALL	13 and neuron\$	68	<u>L4</u>
ALL	12 and adenovirus\$	216	<u>L3</u>
ALL	"free radical\$"	51100	<u>L2</u>
ALL	free radical\$	1677532	<u>L1</u>